THE PHILADELPHIA NEUROLOGICAL SOCIETY.

November 28, 1898.

The President, Dr. F. X. Dercum, in the chair.

Dr. H. C. Wood reported three cases in which pain was an important symptom, and in two of the cases was accompanied by muscular atrophy and paresis. The paper will be published later in this journal.

Dr. Charles K. Mills said that he had had the opportunity to examine the third case with Dr. Wood. He was impressed with the fact that although these three cases occurred in the same family, yet it was not improbable that they did not represent a family type. It was almost impossible to make a diagnosis from the history of the first case, but the record did not seem to permit of it being placed with the other two. While the diagnosis in the third case was difficult, he thought that Dr. Wood's opinion was as nearly correct as any that could be formed. In regard to the second case, he was unable to come to a conclusion. In some of its features it looked like a case of anterior poliomyelitis with pain. The history was peculiar and unlike that of the other two cases.

Dr. William G. Spiller agreed with Dr. Mills that these cases need not be regarded as examples of the same disease. He could not form any idea of the nature of the first case, as the history was incomplete.

The second case, he thought, was probably due to disease of the peripheral neurons. The man had muscular atrophy, reaction of degeneration, loss of muscular power, loss of the patellar reflex in the affected limb, and pain; these are symptoms which point to disease of the peripheral sensory and motor neurons.

The diagnosis in the third case was much more difficult. It might possibly be a very atypical case of amyotrophic lateral sclerosis. The posterior root entrance zones could hardly be much altered. It would be difficult to understand how the reflexes could be exaggerated if these zones were much involved. The reflexes cannot be increased if the sensory portion of the peripheral arc is destroyed, even though the central motor fibers are entirely degenerated. Some of the features in this third case were very unlike those of amyotrophic lateral sclerosis.

Dr. H. C. Wood said that he did not assert that these three cases represented the same disease, but where things are similar it seemed probable that they were due to the same cause. These cases had the three prominent symptoms of pain without tenderness, increasing loss of motility and wasting of the muscles. There must therefore be a suspicion that they were due to the same cause.

He did not believe that simple amyotrophic lateral sclerosis is associated with pain as severe as that which occurred in the third case; he therefore believed that there must be some other lesion present.

A CASE OF PERIODIC FAMILY PARALYSIS.

Dr. J. K. Mitchell presented a patient, the subject of periodic family paralysis. The patient's mother, an uncle, a first cousin, a grandfather and a first cousin of the grandfather, all on the maternal side, had suffered with varying degrees of the same disorder. The patient, aged 18, had his first attack five years ago, since which time they have steadily increased in both frequency and severity, paralysis now occurring about once in eight days, and usually lasting from 36 to 48 hours. He wakes from sleep, unable to move, the disability at first being imperfect, but increasing through the day. During the height of the seizure he can move his head from side to side, but not any limb. All reflexes are lost in severe attacks; plantar, knee, cremaster and abdominal. In the lighter degrees of the palsy the reflexes are slow and small, and very slightly reinforcible. Electric reactions are totally absent in the muscles of the lower limbs in severe, and only producible by very strong galvanic currents in slight attacks. There is during the attack a rough distinct murmur with a splitting of the second sound, heard at the third left costal cartilage, and less clearly along the left sternal margin. The pulse during palsy is sometimes slower than the patient's normal pulse, sometimes faster, and with a good deal of tension. Voice, breathing, swallowing, are unaffected, and the mental faculties perfectly unimpaired. It is sufficient to say without too much detail, that in the intervals the patient is in absolutely perfect health and normal in all respects. Urine, feces and blood have been examined with as yet little result, but this investigation is still in process.

The patient being brought before the society said that since coming to the room he felt the usual warning of an attack in the form of slight general weakness. On attempting to develop his reflexes the knee-jerk was found to be markedly slow and small.

Dr. Charles K. Mills remarked that it seemed impossible to explain such a case as this except on the theory of a temporary toxemia.

Dr. E. Lindon Mellus, of Baltimore, said that the history of this patient suggested to him some of the cases of post-epileptic paresis without epileptic paroxysms.

Dr. E. Lindon Mellus, of Baltimore, read a paper on "Motor Paths in the Brain and Cord of the Monkey." (To be published in this journal.)

Dr. William G. Spiller stated that he wished to report the discovery of a tract not previously described. Some time ago, Dr. Hare reported to this society a case of edema due to a hemorrhage in the external capsule and the lenticular nucleus. The brain was given to Dr. Spiller for examination. In this specimen the latter had found degenerated fibers that certainly came from a level as high as the upper part of the thalamus. They passed down with the degenerated motor tract through the crus, and entered the pons closely associated with the motor tract. A little below the level of the exit of the fifth nerve, a band of fibers became separated from the outermost and lateral portion of the pyramidal tract. Lower down in the pons this band of fibers passed quite abruptly backward and entered the trapezoid body. It took a position at the level of the junction of the medulla oblongata and pons, lateral to the uppermost portion of the inferior olive. As the inferior olive increased in size, this bundle passed backward, and where the olive had its largest width the bundle took a position on the posterior and lateral side of the lower olive. Dr. Spiller had traced this bundle below the motor decussation and had found that it remained on the same side throughout its entire length. He had followed this band of fibers from the level of the upper part of the lenticular nucleus as far as the first cervical segment. In this specimen he had also traced motor fibers to the lateral tract on the same side of the cord, as Dr. Mellus had done in the monkey.

Dr. Spiller agreed with Dr. Mellus in the statement that the failure to stain the degenerated collaterals of the pyramidal fibers within the anterior horns might be due to the fact that these fibers are so small that the degenerated myelin is not very distinct; he did know, however, that the medullary sheath remains on the reflex collaterals of the posterior roots after these collaterals enter the gray matter. Dr. Dejerine and he had been able to trace collaterals of the posterior roots into the anterior horns.

The black dots which Dr. Mellus had shown in the perivascular spaces and had regarded as contained within the cells lining these spaces, Dr. Spiller had seen a number of times, and believed that they were contained within the granular bodies, the "corps granuleux" of the French writers.

December 19, 1898.

Dr. Charles K. Mills in the chair.

A CASE OF CEREBELLAR TUMOR.

Dr. Spiller presented a woman of 43 years who began to have severe headache four years ago. The headache lasted two years, then disappeared, and during the last few weeks had returned over the left side of the head behind and above the ear. This region was very sensitive to the slightest touch. The woman had had extreme vertigo during the past two years. She could lie on her right side, *but lying on her left side caused unendurable vertigo.* She had not had vomiting, disturbance of the sphincters, or pain in the limbs. The gait was extremely cerebellar in type and Romberg's sign was pronounced. The patellar reflex was absent on each side, and tactile sensation below the knees was diminished. A tendency to fall backward was very evident. The reaction of the pupils was normal, and the cranial nerves were not affected. The optic disks were somewhat congested. A history of a discharge of pus from the left ear two years ago was given, and Dr. Randall found healed perforation of each ear. No word-deafness, no word-blindness, no hemianopsia, and no mental disturbance existed.

Dr. Spiller regarded this case as one of cerebellar disease, probably tumor or abscess situated in, or pressing upon, the cerebellum; although dilatation of the fourth ventricle or meningitis might cause such a symptom-complex. The only localizing symptoms were the extreme vertigo when the patient laid upon her left side, and the great sensitiveness of the region behind and above the left ear. It was extremely difficult, therefore, to suggest to the surgeon the proper place for operation. The sensitive area was indicative of a lesion in the lower part of the left occipito-temporal lobe; the extreme vertigo when the patient laid upon her left side was suggestive of a tumor in the right cerebellar hemisphere, causing pressure upon the cerebellum when the right side of the body was uppermost.

Dr. Charles K. Mills thought that this case was one either of tumor or abscess of the cerebellum, with the probabilities in favor of tumor. The pain was not over the cerebellum but over the occipital lobe, and in the neighborhood of the parieto-occipital fissure. The patient, however, did not have the distinctive symptoms of tumor or abscess involving the left occipital lobe. There was no word-blindness and no hemianopsia. The position of the pain would seem to indicate that the tumor was separated anatomically from the cerebellum. He thought possibly that some pachymeningitis might be associated with the cerebellar tumor. As the case was a serious one and progressing, he believed that an exploratory operation was advisable.

In the study of such cases as this, Dr. Mills said it was of interest to study not only the quadriceps-jerk and knee-jerk, but also the gastrocnemius-jerk and ankle-jerk. In the present case, the muscle and tendon phenomena in the lower extremities were entirely absent. He had recently studied the gastrocnemius-jerk and ankle-jerk in a number of cases of tabes. About twenty-eight cases had been examined, and in all but three the tendo-Achillis jerk was absent. In one case it was present only on one side. In one case it was rather plus. The gastrocnemius-jerk was present in all. This matter might be of some importance in the differential diagnosis of a case of the kind presented by Dr. Spiller in its early stage from a case of tabes. In this patient with cerebellar tumor there was loss not only of the patellar reflex and Achilles-jerk, but also of the gastrocnemius-jerk and quadriceps-jerk. In almost every case of tabes which he had examined in this connection, the quadriceps-jerk and gastocnemius-jerk were preserved.

Dr. Spiller remarked that the question of the absence of the reflexes in brain tumor is one of great interest and one that we understand very imperfectly. In a certain number of these cases examined post mortem, degeneration of the posterior roots had been found. Numerous papers have been written to show why this occurs. It is held by some that the cachexia causes the degeneration of the posterior roots. By others the theory is held that this posterior root degeneration is due to pressure; that there is an increase of the cerebrospinal fluid and that the pressure is exerted especially in the lower part of the cord. Those who oppose this theory bring forward the statement that the degeneration may involve the cervical cord more than the lumbar, and contend that the pressure theory is not satisfactory.

FIVE CASES OF TABES IN THE NEGRO.

Dr. J. W. McConnell stated that about six years ago in concluding a paper read before this society, Dr. Burr had said that "while the data given are not sufficient to permit one to say dogmatically that locomotor ataxia is a rare disease among negroes, still they are sufficient to permit one to hold such an opinion tentatively until further investigation disproves it." It was not Dr. McConnell's intention to gainsay the opinion expressed by Dr. Burr at that time, but rather to emphasize it and to add these cases to those now known.

Eight years' service at the dispensary for nervous diseases at the Philadelphia Polyclinic had given him an opportunity to see many cases of tabes in the white race, quite a few among the mixed races, and but five instances of the disease in pure negroes.

Case I.—B., full blooded negro, age 55 yrs., native of Virginia, a sailor by occupation. Earliest symptoms appeared fifteen years ago; sharp, cutting pains in legs and thighs, failure of sight in left eye, two years later right eye became involved. Condition grew progressively worse. Specific history acknowledged. Examination showed no ataxia, no Romberg sign, no paralysis or paresis. Total blindness, pupils contracted and immovable to light stimulation. Ophthalmologist reported a condition of primary optic atrophy in both eyes. Knee-jerks retained, but diminished.

Case II.—G., full blooded negro, parents born in Liberia, age 49 yrs. Twelve years ago had a chancre followed by secondaries. Weakness in the legs for ten years, unsteady on his feet, shooting pains in both lower extremities. Has had girdle sensation, also a feeling as of a veil over his face. At present he is totally blind in the right eye, partially so in left. Typical primary degeneration of both optic nerves. Knee-jerks absolutely lost. He has sharp pains in legs, numbness of the right hand and of the ulnar edge of the left hand. No rectal or vesical trouble.

Case III.—L., negro waiter, born in Virginia, aged 44 years; specific history. Pain in legs for some years, with some difficulty in walking. Lately failing sight. Knee-jerks lost; slight sway, very slightly ataxic gait. Pupils small, disks show advancing gray degeneration.

Case IV.—W., aged 51 yrs., negro born in North Carolina; specific infection, twenty-four years ago, secondaries quite free. Pain in legs commenced fifteen years ago. Diplopia about ten years ago, and soon after failure of vision. At present the patient is almost blind in both eyes, has very small pupils. He is not ataxic, does not sway abnormally. Knee-jerks are abolished. He has difficulty in holding his water and also in controlling the sphincter ani.

Case V.—T., female, full blooded negress, aged 34 (?) years, native of Virginia, married. Possible specific infection ten years ago. Sight has been failing for two years in the right eye, for one year in the left. At present, ataxia of lower extremities, sway increased, sharp pains in legs, girdle sensation, feeling as of a cobweb over her face, difficulty in holding water. Knee-jerks absolutely lost. Is almost blind in both eyes and ophthalmologist reports complete spinal atrophy of both optic nerves. Absolute iridoplegia.

Some features of the cases were worth emphasis.

Four of the patients were men and one a woman. All five were pure negroes. The parents of four were known by the respective patients to have been negroes, and one of these four was the son of parents born in Liberia. The fifth had been a slave, the son of a negro slave father. He did not recollect ever having seen his mother, but as the man was very black Dr. McConnell presumed that his mother was a negress.

The cases were all of the amaurotic form of tabes. In three instances the failure of vision was gradual and came on after the pains, and affected both eyes simultaneously and in like degree. In the other two the vision of one eye failed before that of the other. Four were almost blind, although one patient was relatively less so than the others. The fifth was reported by the ophthalmologist as "one of pallid disks which were very suggestive of advancing tabetic atrophy." Three of the patients were distinctly ataxic in the lower extremities; the woman had also some difficulty in using the upper extremities. In these three the Romberg sign was obtainable.

The woman and one of the men presented many of the usual text-book symptons of tabes dorsalis. Two of the other three suffered with lancinating pains, the knee-jerks were abolished, the irides were not responsive to light, the optic nerve was the seat of tabetic atrophy. One of the two had Romberg's symptom. A fifth patient, most blind of all, had shooting pains and girdle sensation, but no ataxia and no Romberg's sign. He had retained the knee-jerks. These were perhaps not as prompt as in a normal individual, but they were present. In all five there was iridoplegia. Two cases had some involvement of the vesical sphincter; two complained of a feeling as if the face was covered with cobwebs or a veil. One case presented numbness of the ulnar edge of the left hand and of the whole of the right hand.

The four men admitted specific infection, and in the woman it was very probable from her own statements.

Dr. William G. Spiller said that three of the cases had come to the Polyclinic Hospital since he had been connected with the service. They were all of the amaurotic type. If the negro is more liable to this form of tabes, it may explain why the disease is considered uncommon in the negro, inasmuch as the amaurotic form is at times overlooked by the general practitioner. A year ago, he presented one or two patients with the amaurotic form, and at the time spoke of Edinger's substitution theory, and said that this theory did not explain the amaurotic form. If the disease occurred in those who used their eyes to excess we might understand why overwork has something to do with it. He had recently received a letter from Prof. Edinger in which the latter, in the spirit of a true scientist, acknowledged that his theory would not explain every condition in tabes, but stated that it explained much better than any other so many symptoms that it must contain a "fragment of truth." Prof. Edinger also spoke of the great importance of the statements regarding the resistance of the negro to tabes. Dr. Spiller referred to the exceedingly interesting experiments which Prof. Edinger had performed on rats.

Dr. John K. Mitchell said that Dr. Burr seemed to assume the almost necessary presence of syphilis in these cases. While he recognized the frequency with which syphilis precedes tabes, yet the more he saw, the more he doubted the supposed necessary relation between the two. A careful lookout for tabes in the negro had been kept at the Orthopedic Hospital for twelve or thirteen years, and not more than three cases had been found; one of these was in a very light mulatto.

He thought that the blindness might result in a certain concealment of some of the tabetic symptoms: the blind patient, if he can walk at all, necessarily does so without seeing his feet; thus perforce he acquires some of the greater ease of motion and improvement in station which tabetics acquire by learning to execute careful coordinate movements—not that they have changed the diseased tissues of the cord, but that they use themselves enough better to conceal some of the symptoms of the disorder.

Dr. Burr replied that he did not belive that syphilis is the only cause of tabes dorsalis

Dr. Joseph Sailer reported a case of hemiplegia with astereognosis.

Dr. J. T. Rugh reported a case of hysteria simulating ascending neuritis. (To be published later in this journal.)

Dr. F. Savary Pearce said he believed this patient had neuritis with hysteria.

Dr. Spiller had seen this patient in consultation and had found a very serious condition of the left arm. The arm was much swollen. The patient felt tactile sensation at every part of the arm; when a pin was thrust into the skin she felt it as touch and not as pain; she had, therefore, dissociation of sensation. Knowing the great rarity of ascending neuritis, especially in the absence of pus formation, Dr. Spiller was exceedingly skeptical as to its presence in this case. The electrical reactions were found to be perfectly normal, except that a stronger current was required on account of the swelling. Within twenty-four hours after Dr. Spiller first saw the patient the paralysis had become complete and sensation was lost from the shoulder down. The pain, which had been intense, had entirely disappeared. He was convinced that hysteria was the cause of this apparent neuritis. A purely suggestive treatment was adopted and in three or four days all the signs of the supposed neuritis had disappeared.

33. UN CAS DE PARALYSIE GÉNÉRALE JUVENILE AVEC SYPHILIS HÉREDITAIRE (A Case of Juvenile General Paresis, with Hereditary Syphilis). A. Saporito (Ann. méd. Psychol., 6, 1898).

General paresis developed in this young man at the age of 18. There was no particular neuropathic nor psychopathic heredity, but there were evident signs in the patient of hereditary syphilis. He was poorly developed, had an infantile head with Hutchinson teeth and other characteristic marks. He was always somewhat peculiar, but was comparatively bright up to the age of 16, when a gradual dementia began with general elevation of the ego. There were typical changes in his speech, fibrillary tremors of face and tongue, Argyll Robertson pupils, with inequality, epileptoid attacks, marasmus and death. The pathological findings were those usually described as resulting from general paresis.